SHORT REPORT

# Effects of decompressive craniectomy on brain tissue oxygen in patients with intracranial hypertension

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This report examined the intraoperative course of partial pressure of brain tissue oxygen ($P_{ti}O_2$) and intracranial pressure (ICP) during surgical decompressive craniectomy for medically intractable intracranial hypertension due to diffuse brain swelling in three patients after severe subarachnoid haemorrhage and aneurysm coiling. The mean ICP decreased from 59 mm Hg to 10 mm Hg in a two step fashion, relating to bone flap removal and dural opening. Simultaneously, $P_{ti}O_2$ increased rapidly from 0.8 kPa (6 mm Hg) to 3.07 kPa (23 mm Hg). $P_{ti}O_2$ and ICP remained at non-critical ranges postoperatively. Despite these beneficial effects on ICP and $P_{ti}O_2$, the patients' clinical status remained poor with two in a persistent vegetative state and one dead.

Control of increased intracranial pressure (ICP) remains an important challenge in the treatment of patients with severe post-stroke or post-traumatic brain oedema. Decompressive craniectomy has been proposed as an effective treatment as beneficial effects on outcome have been reported in clinical trials on patients with traumatic brain injury and middle cerebral artery stroke.[1–6] Anecdotal reports and small series suggest decompression may also be successful in other diseases associated with high ICP, such as encephalitis, metabolic encephalopathy, and subarachnoid haemorrhage (SAH).[7–10]

There exists little information about the pathophysiological changes induced by the cranial decompression. To gain more information about these effects, we investigated the intraoperative course of ICP and partial pressure of brain tissue oxygen ($P_{ti}O_2$) during surgical decompression in three patients with medically intractable intracranial hypertension, occurring after severe aneurysmal SAH. The monitoring of $P_{ti}O_2$ with the polarographic Clark-type probe has been proven to be a reliable tool for detecting cerebral hypoxic events after severe cerebral insults.[11–14] The estimated threshold for significant cerebral tissue hypoxia is reported to be at about 1.33 kPa (10 mm Hg). Values below this threshold indicate critical cerebral oxygenation and a high risk of secondary brain damage.

## PATIENTS

Three patients suffering from severe cerebral oedema and intracranial hypertension after aneurysmal SAH were studied. Clinical data are given in table 1. External ventricular drains for haemorrhagic hydrocephalus were placed in all patients after admission and the aneurysms were coiled within two days of SAH. Thereafter, all patients developed increased intracranial pressure because of diffuse brain swelling refractory to medical treatment, including analgesia, sedation, mannitol, hypertonic saline, TRIS buffer (THAM), moderate hyperventilation ($P_aCO_2$ about 4.67 kPa (35 mm Hg)) and barbiturate coma.

ICP probes (Codman and Shurtleff, Raynham, MA, USA) and $P_{ti}O_2$ probes (LICOX Systems, GMS mbH, Kiel, Germany) were inserted into the cerebral white matter via a double lumen bolt located about 15 mm lateral to midline and 20 mm anterior to the coronal suture. $P_{ti}O_2$ probes were placed into CT viable tissue at a depth of 22–27 mm in the anterior cerebral artery vascular territory, as this was initially considered to be tissue at risk for development of symptomatic cerebral vasospasm. Neuromonitoring started 4 hours, 46 hours, and 33 hours after the haemorrhage. To observe the immediate effects of decompression, data collected intraoperatively of ICP and $P_{ti}O_2$ were stored on a computer with a rate of 6/min. Mean arterial pressure was monitored via a radial artery catheter referenced to the foramen of Monro.

Decompressive fronto-temporo-parietal craniectomy (diameter about 12 cm) was performed after medical treatment failed to keep ICP values below 30 mm Hg. Preoperatively, the implanted probes were meticulously covered with a sterile dressing to avoid contamination of the surgical field. After the removal of the bone flap, the dura was opened to provide maximum reduction of ICP and duraplasty with periosteum was performed.

## RESULTS

Before removal of the bone flap patients exhibited hypoxic mean $P_{ti}O_2$ values of 0.8 kPa (6 mm Hg) and mean ICP of 59

**Abbreviations:** ICP, intracranial pressure; $P_{ti}O_2$, partial pressure of brain tissue oxygen; SAH, subarachnoid haemorrhage

**Table 1** Clinical data of three patients undergoing intraoperative monitoring of ICP and $P_{ti}O_2$ during decompressive craniectomy

| Case | Age (y), sex | WFNS on admission | Aneurysm location | Side of Probes | Side of decompression | Decompression (days after SAH) |
|---|---|---|---|---|---|---|
| 1 | 60, F | 5 | A-com-A | Left | Left | 5 |
| 2 | 36, F | 5 | ICA left | Left | Left | 2 |
| 3 | 36, F | 4 | ICA right | Right | Right | 2 |

A-com-A, anterior communicating artery; ICA, internal carotid artery; SAH, subarachnoid haemorrhage; WFNS, World Federation of Neurosurgeons grading scale for SAH.

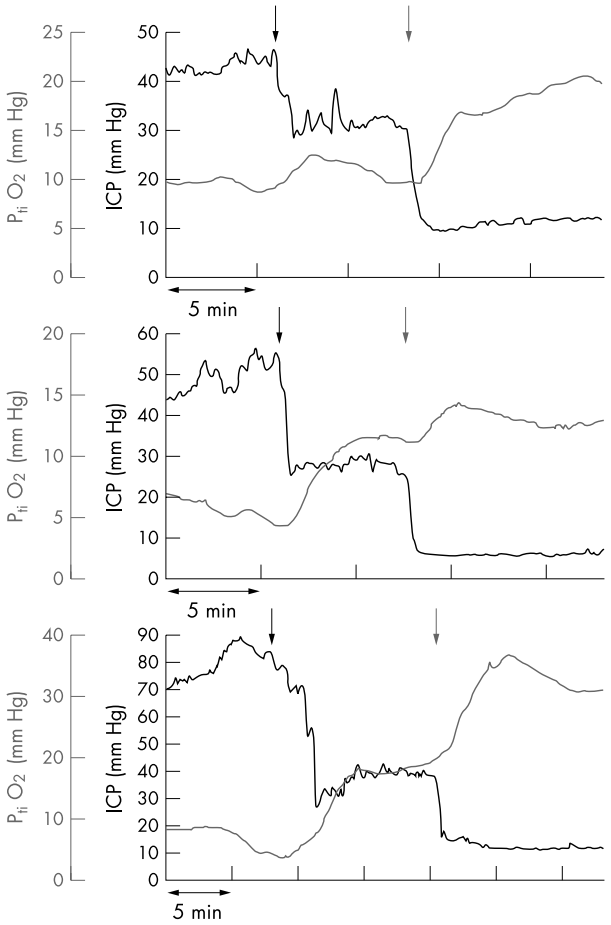


**Figure 1** Three graphs demonstrating the intraoperative course of ICP and $P_{ti}O_2$ for patients number 1, 2, and 3. Black lines indicate ICP, grey lines $P_tO_2$. The removal of the bone flap is indicated by the first arrow (closed tip), opening of the dura by the second arrow (open tip).

mm Hg. The individual intraoperative course of $P_{ti}O_2$ and ICP for each patient is shown in figure 1. The immediate two step reduction of ICP to 32 mm Hg after removal of the bone flap and to 10 mm Hg after opening of the dura was accompanied by a simultaneous improvement of $P_{ti}O_2$ above hypoxic thresholds to 3.07 kPa (23 mm Hg). During the procedures mean arterial pressure was stable between 100 mm Hg and 120 mm Hg in all three patients. $P_aO_2$ was kept at about 16 kPa (120 mm Hg) and frequently checked by arterial blood gas measurements. In the postoperative course $P_{ti}O_2$ and ICP constantly remained at non-critical ranges. Routine CT scan obtained at the first postoperative day excluded cerebral infarction and showed the maintained correct position of the implanted probes. Critical cerebral vasospasm at the time of decompression was unlikely, as preoperative and postoperative transcranial Doppler measurements revealed blood flow velocities below 120 cm/s. At six months after the haemorrhage, patients 1 and 2 remain in a persistent vegetative state and patient 3 died. No wound infections occurred in the postoperative period.

## DISCUSSION

The results of these three cases of intraoperative $P_{ti}O_2$ monitoring demonstrate that the immediate reversal of critical cerebral oxygenation is possible with the use of decompressive craniectomy. In all these patients suffering from severely raised ICP and diminished cerebral perfusion pressure, $P_{ti}O_2$ rapidly increased to non-hypoxic levels. The simultaneous dramatic two step reduction of raised ICP to normal values during removal of the bone flap and dural opening has been described in a similar pattern by Yoo *et al*,[15] however, our data for the first time provide evidence of immediate positive effects on cerebral tissue oxygenation in humans. Previous clinical reports suggest that the decompression related increases in $P_{ti}O_2$ are predominantly induced by simultaneous increases of cerebral blood flow. With the rapid normalisation of cerebral perfusion pressure, both transcranial Doppler and single photon emission computed tomography studies were able to show that the depressed cerebral circulation improved after the procedure.[16 17] Furthermore, during evacuation of acute subdural haematomas, rapid increases of laser Doppler flow have been found, indicating improved cerebral blood flow and oxygen delivery.[18]

Despite the demonstrated positive effects of surgical decompression on cerebral oxygen content, the rapid and successful treatment of low $P_{ti}O_2$ and raised ICP did not translate into improved outcome with two patients remaining in a persistent vegetative state and one dead. The beneficial effects on cerebral oxygen content and ICP because of decompression were most probably offset by the devastating primary brain damage attributable to the initial haemorrhage and the natural course of such severe primary irreversible loss of neuronal function. In addition, ICP and $P_{ti}O_2$ were in highly abnormal ranges at the time of intervention, making secondary brain damage very probable.

As well as post-SAH swelling, we are currently expanding our study to include post-traumatic patients. However, difficulties arise because the timing (early compared with "last option") of the surgical decompression may well change the pathophysiological responses. Based on the generally accepted knowledge that high ICP and low $P_{ti}O_2$ levels are important contributors to a poor outcome, the data presented favour the early use of decompression, particular if metabolic monitoring is being carried out. Our experience suggests, however, that extended neuromonitoring with intracranial probes via a multichannel skull bolt during decompression is a feasible method for intraoperative evaluation of neurometabolic parameters.

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# NEUROLOGICAL STAMP

## Fridtjof Nansen (1861–1930)


Little is written in textbooks of medical history about Nansen, who is better known as the Norwegian who founded modern polar exploration. His contributions were in many spheres. Nansen was an invertebrate zoologist who in 1882 was appointed curator of zoology at the Bergen museum. He stayed in Bergen for 5 years, focusing his interests on the neuroanatomy of marine invertebrates. For one of his papers "The structure and combination of histological elements of the central nervous system" (1887), the university in Kristiana conferred upon him the degree of doctor of philosophy. His dissertation contained so many novel interpretations that the examination committee accepted it with reluctance, but the work is now considered a classic. Two days after his dissertation was accepted Nansen was on his way to Greenland. He crossed Greenland on skis during 1888–1889. Nansen was appointed professor of zoology at the University of Oslo in 1887 and in oceanography in 1908. On the basis of his research on the nervous system of an obscure marine invertebrate, the myzostome, he first expressed doubt about the reticular nature of nervous structure. In 1886 he visited Camillo Golgi at the University of Pavia and learnt Golgi's new method of staining nerve cells. Nansen was preoccupied with the question of how nerves communicate with each other and was a pioneer advocate of what later became known as the neurone doctrine. He was quite adamant that nerve units were not fused, but only touched each other. Nansen, His, and Forel, working from different points of view, had sown the seeds of doubt about the reticular theory and became cofounders of the modern view of the nervous system. Ramon Y Cajal's declaration of the independence of the nervous cellular unit was published in May 1888, a year after Nansen's original paper. Nansen also offered an explanation of the reflex arc, proposing that sensory nerves conduct information from the periphery, and central cells relay the impulses to motor nerves, which activate muscles. He discovered that spinal ganglia bifurcate into ascending and descending processes, and was the first to postulate the ectodermal origin of Schwann cells. Nansen also described Leydig's "dotted substance", now called the neuropil, and showed that the nerve fibres in the dotted substance were in contact, but did not anastomose with each other. By 1906, when Raymon Y Cajal and Golgi were sharing the Nobel Prize in physiology or medicine, Nansen was Norwegian ambassador in London. He had become famous for his exploration of the Arctic, and had played a key part in the dissolution of the union between Sweden and Norway. His endeavours were probably decisive in avoiding war and ensuring peaceful collaboration between Norway and Sweden. Later, he made major contributions to the foundation of the science of physical oceanography, and after the First World War worked extensively with the repatriation of prisoners of war and refugees, and with famine relief. For his humanitarian efforts he was awarded the Nobel Peace Prize in 1922. He is shown here in one of his several philatelic honourings on a stamp from Norway with Roald Amundsen, the Norwegian explorer who was first to reach the South Pole on December 14 1911, 33 days before Scott (Stanley Gibbons no 392, Scott no 287). When Amundsen left Norway he sailed in Nansen's old ship *Fram* (Norwegian for Forward), which is also shown. *Fram* was a unique vessel designed for Nansen to resist the extreme pressures of pack ice in the polar regions. Amundsen had studied medicine for period but withdrew to go to sea.

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